recovery of renal function with resolution of pulmonary oedema.

COMMENT

In elderly diabetic patients with renal cholesterol atheroembolism, a common precipitant is manipulation of atherosclerotic vessels during vascular surgery or angiography. It can also result from thrombolytic and anticoagulant therapy, sometimes after a considerable delay[1]. Spontaneous cholesterol embolism is uncommon—found by Cross[2] in 1.9% of serial necropsies, always in patients over 60 years of age. The clinical features are varied and make diagnosis difficult. Risk factors for renal cholesterol atheroembolism are advanced age (mean 66 years), hypertension, coronary atherosclerosis and renal impairment[3]. Spontaneous renal atheroembolism often leads to progressive decline in renal function, early dialysis-dependence and high mortality[3]; however, renal function can recover[4]. Flash pulmonary oedema often points to underlying atheromatous renal artery occlusion, and we suspect that this was present in our patient. Regarding treatment, there is some evidence that a statin can stabilize atherosclerotic plaques, reduce the propensity for atheroembolism and thus preserve renal function[5].

Recurrent spontaneous cholesterol atheroembolism, characterized here by short-lived episodes of acute renal failure and pulmonary oedema, does not seem to have been described previously. This possibility should be considered in any elderly diabetic patient with established atherosclerotic disease who presents with impaired renal function and pulmonary oedema.

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Systemic vasculitis with multiple aneurysms complicating systemic lupus erythematosus

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Although immune complex deposition in and around small arteries is a common finding in patients with systemic lupus erythematosus (SLE), severe acute inflammation of the arterial wall is seldom described in the condition. By way of contrast, the characteristic histological changes in polyarteritis nodosa and Wegener's granulomatosis include acute inflammatory lesions of the arteries in the absence of immune complex deposition[1]. Here we present a case where classic SLE evolved into a systemic vasculitis, with multiple aneurysm formation.

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CASE HISTORY

A woman aged 24 was seen at a district hospital with arthritis affecting shoulders, hands, and knees. The initial investigations revealed lymphopenia, a mild normochromic normocytic anaemia and an erythrocyte sedimentation rate (ESR) of 104 mm in the first hour. She was found to have a raised titre of antibody against double-stranded DNA, Ro, La, and Sm. SLE was diagnosed and she was started on treatment with a non-steroidal anti-inflammatory drug, but joint pains remained troublesome. Six months later she developed mild oedema and her urine contained protein and a trace of blood. Protein excretion was 0.9 g per 24 hours and creatinine clearance was 142 mL/min. After a further 2 years, low-dose prednisolone was prescribed for symptomatic relief of pleurisy and joint pains. 3 years after the onset of disease she developed acute onset epigastric pain, and abdominal ultrasound demonstrated a vascular lesion in the liver. She was referred to the department of surgery in this hospital.

On admission she complained of a constant severe epigastric pain radiating to the back. There had been no vomiting or anorexia. The pain was not exacerbated by movement or posture. Medication on admission was prednisolone 5 mg per day, omeprazole 20 mg per day

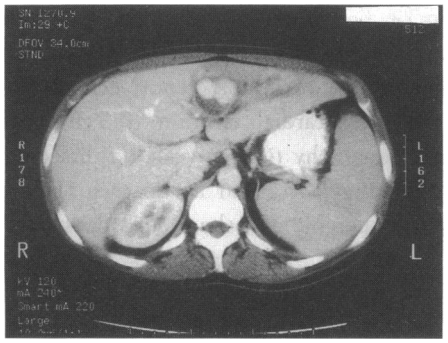

Figure 1 **Spiral computed tomographic scan of liver showing contrast-enhancing mass in left lobe**

and amlodipine 10 mg per day. On examination there was no cutaneous vasculitis, alopecia or synovitis. She was apyrexial and normotensive. The only abnormal finding on abdominal examination was mild epigastric tenderness on deep palpation. The chest and cardiovascular system examinations were normal. Periorbital oedema was present. The urine contained protein (++) but was free from blood and the sediment was bland. Initial investigations were haemoglobin 10.3 g/dL, white cells 9.1×10^9/L (lymphocytes 0.8), platelets 460×10^9/L, ESR 92 mm/h, urea 5.6 mmol/L, electrolytes and creatinine normal, alkaline phosphatase 254 IU/L (normal 130), other liver enzymes and bilirubin normal, C reactive protein 81 mg/L (0–5).

A spiral computed tomographic (CT) scan of the abdomen with intravenous contrast revealed a vascular lesion in the left lobe of the liver (Figure 1), and an arteriogram (Figure 2) showed multiple aneurysms of the hepatic arteries and a large false aneurysm from a segmental artery in the left lobe of the liver. To occlude the feeding vessel of the aneurysm, a coil was placed transarterially. A further angiographic study revealed multiple small aneurysms of the splenic and gastroduodenal arteries, a 5 mm aneurysm in the mid pole of the right kidney, and a 2 mm aneurysm in the upper pole of the left kidney. Blood testing showed a persistent acute phase response; she was seronegative for antineutrophilic cytoplasmic antibodies and positive for rheumatoid factor (1/1000), anti-DsDNA, and anti-Ro, La, and Sm. C3 complement was 138 mg/dL (<70), and C4 complement was 19 mg/dL (>16); there was no evidence of previous infection with hepatitis B.

She was given intravenous methylprednisolone 500 mg on each of three consecutive days and the dose of oral prednisolone was increased to 60 mg per day. Monthly pulsed cyclophosphamide therapy was then begun, with tapering of the prednisolone to 25 mg per day. After three months of follow-up she remains well and the acute phase response has settled. A follow-up CT scan shows resolution of the false aneurysm.

COMMENT

This patient with classic SLE developed a systemic vasculitis with features of polyarteritis nodosa. Several case reports

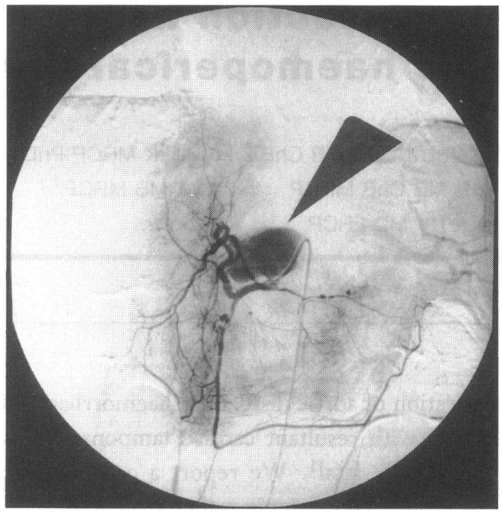

Figure 2 **Arteriogram showing multiple aneurysms and large false aneurysm in left lobe of liver**

describe the development of a solitary aneurysm in the setting of SLE[2–4], but progression of SLE to systemic vasculitis has been reported only as a late complication, appearing more than 10 years after the onset of disease[5].

One possible explanation for this apparent association is that the initial diagnosis of SLE was erroneous and the clinical features at that stage were due to systemic vasculitis. However, this patient satisfied commonly used criteria for the classification of SLE[6] well before presentation with vasculitis. Further possibilities include the development of two unrelated conditions, or a common pathogenic factor causing progression from SLE to systemic vasculitis. The exposure of arterial antigens by SLE-associated tissue damage or the production of anti-endothelial cell antibodies in the context of SLE might account for the progression of the disease in this way.

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